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Amino acid substitution at position 226 of the hemagglutinin molecule of influenza (H1N1) virus affects receptor binding activity but not fusion activity.

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The receptor binding site of the hemagglutinin (HA) molecule of type A influenza virus A/USSR/90/77 (H1N1) has been studied. Site-specific mutagenesis has been used to introduce base changes into the sequence that codes for the amino acid residue at position 226 on the HA molecule, and mutant sequences replaced the wild-type sequence of the HA gene of the SV40-HA recombinant virus (SVHA). Mutant HA proteins were expressed in African green monkey kidney cells and analyzed for receptor binding and fusion activities. Two mutant HA proteins containing single amino acid substitutions of Asn and Met for Gln at position 226 retained their receptor binding activity, but others with amino acid substitutions Glu, His, Leu, Val, and Thr for Gln at position 226 lost this activity. All the mutant proteins retained their fusion activity. On the other hand, another four mutants containing single amino acid substitutions at positions other than 226 retained the receptor binding and fusion activities, despite the drastic change in charge or polarity to the respective amino acids. These results suggest that amino acid residue 226 of the H1 subtype of HA is critical for receptor binding activity of the HA protein. Our results also show that the lack of receptor binding activity of the HA protein does not affect fusion activity.

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